Technical Bulletin 307 – 1976

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Cooperative investigations with the Agricultural Experiment Stations of the University of Minnesota, Colorado State University, the University of Puerto Rico, and the U.S. Department of Agriculture. The work was supported in part by a grant from the U.S. Department of Agriculture, ARS Cooperative Agreement 12-14-100-10-046 (34), to the University of Minnesota.

We thank Dr. R.W. Romig, formerly of the Cereal Rust Laboratory and now with Northrup King and Co., for his help with the early planning of this work and for his continued interest and support.

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INTRODUCTION

In this bulletin, we describe a comprehensive study of yield losses in spring wheat due to the wheat stem rust disease, incited by *Puccinia graminis* Pers. f. sp. *tritici* Eriks. & E. Henn. The main objective of the study was to develop a model that would improve the accuracy of estimates of yield losses due to wheat stem rust. A three-dimensional graphic model that related disease epidemic to crop loss was created from a large body of experimental field data. The studies of other scientists are reviewed, and the basic concepts of the disease and yield measurements are discussed. We describe the design of the field experiment, report its results, and finally, explain the development of the new model, its strengths and possible weaknesses, and discuss the relation of the model to the concepts of disease resistance and tolerance in plants.

The need for accurate, reliable methods to determine yield losses will increase as the increasing world population creates additional demands for food. Efficient food production, which has become essential, requires control of avoidable losses in yield. Losses from most infectious plant diseases are often difficult to measure, except when the yield is completely destroyed. For example, loose smut of wheat destroys the entire head and the percentage of yield loss can be counted directly by observation. More commonly, the diseased plant produces a harvestable crop and the amount of yield loss is obscured.

Reliable methodology to estimate crop losses could enable planters to weigh the cost of a control program against the benefits of increased yield, and thus could play a key role in profitable crop management.

A method for estimating crop losses from disease could have several functions. It could help farmers predict potential losses so that control measures could be initiated to prevent losses. The method could reveal the loss too late to initiate control measures but early enough to enable the farmer to estimate the size of the ultimate crop and to base shipping and marketing decisions on his estimations. The method could help provide *ex post facto* estimates of the loss, information that could be useful for evaluating the economic importance of the disease (Chester, 1950).

The disease-biological system studied, stem rust on spring wheat, was chosen because wheat is the world's most widely cultivated crop, and stem rust frequently infects wheat. Furthermore, wheat and rust are both convenient experimental materials for which a considerable background of knowledge and technique was available. The crop is an annual and the stem rust is relatively easy to observe.

During the past 45-50 years, numerous attempts have been made to develop a system for measuring cereal losses due to rusts (e.g. Kirby and Archer, 1927; Chester, 1950). No system has been accepted generally by persons associated with wheat production. To be widely accepted, a system that relates wheat loss to disease should be based upon data from several genotypes that grow in several environments and have different levels of disease. Loss measurements should be as accurate as possible. Previous studies involved one or more but not all of these factors. Therefore, this field experiment was designed to encompass these factors and to provide a large amount of data from which could be constructed a generalized model that would relate disease to loss. The wheat genotypes, environments, and disease levels were the dependent variables, while the fungus genotype was held constant.

Several approaches have been suggested for developing a model for estimating yield loss from disease data (James, 1974). One experimenter related loss to the area under the disease-progress curve (Van der Plank, 1963); he presumed that the area was proportional to loss. This concept applied to curves with similar origins but different areas, but did not seem to operate for curves with different origins but equal areas (Romig and Calpouzos, 1970). Other scientists related loss to a critical stage of host growth; they theorized that the stress of disease at a critical physiological growth stage of the host was proportional to the loss (Romig and Calpouzos, 1970). That relationship must apply in some instances, but general application might be questioned. Nevertheless, scientists should consider the growth stage of the host when measuring disease incidence. Still another approach encompassed both the area under the curve and the critical stage methods; at each plant growth stage, disease incidence was assigned an additive value that was related to yield loss (James *et al.*, 1972).

A good method for measuring disease losses should be relatively simple, accurate, and reliable. The method should be represented as a model either as a mathematical formula, table, or graph. Some of these alternatives were considered in this study and are described in detail in the following sections.

MANIPULATING AND MEASURING STEM RUST EPIDEMICS

Measuring stem rust development: Only methods of estimating rust severity were reviewed because the methods for estimating disease in general were reviewed already by Large (1966) and Chester (1950). Cobb (1892) developed a scale with diagrams for estimating the area occupied by rust pustules on the flag leaf and the second leaf of the wheat plant. The diagrams illustrated 1, 5, 10, 20, and 50 percent of the leaf area occupied by rust pustules. This method was modified by Melchers and Parker (1922) who determined that rustiness was maximum ("100 percent severity") when only 37 percent of the actual area of the leaf or stem was covered with rust. Their diagrams illustrated 5, 10, 25, 40, 65, and 100 percent severity. Peterson, Campbell, and Hannah (1948) thought the systems of Cobb and of Melchers and Parker were inadequate because: "1. Too few diagrams are shown, making a great deal of interpolation necessary when recording rust readings in intervals of 5 to 10 percent. 2. The irregular intervals used make classification difficult. 3. The depicted pustules do not adequately represent the great range in size of uredia and telia of rusts occurring on cereals." Therefore, they presented diagrams to indicate that pustules of different numbers and sizes could give similar rust severity ratings. They distinguished among 12 categories of rust severities ranging from 1 to 100 percent.

The systems of Cobb, of Melchers and Parker, and of Peterson, Campbell, and Hannah were based on the proportion of surface area of the host that was covered with rust pustules and permitted rapid and consistent estimation of rust severity. Because the system of Peterson, Campbell, and Hannah seemed more flexible than the other two systems, we adopted it for our work (figure 1).

Kingsolver *et al.* (1959) also refined the Cobb system by counting uredia on 20 successive culms from different places in the plots. When it became impractical to count the individual uredia, they estimated that 10 pustules per culm were equivalent to 1 percent rust severity.

Recently Burleigh, Romig, and Roelfs (1969) demonstrated that the severity of epidemics of leaf and stem rusts of wheat

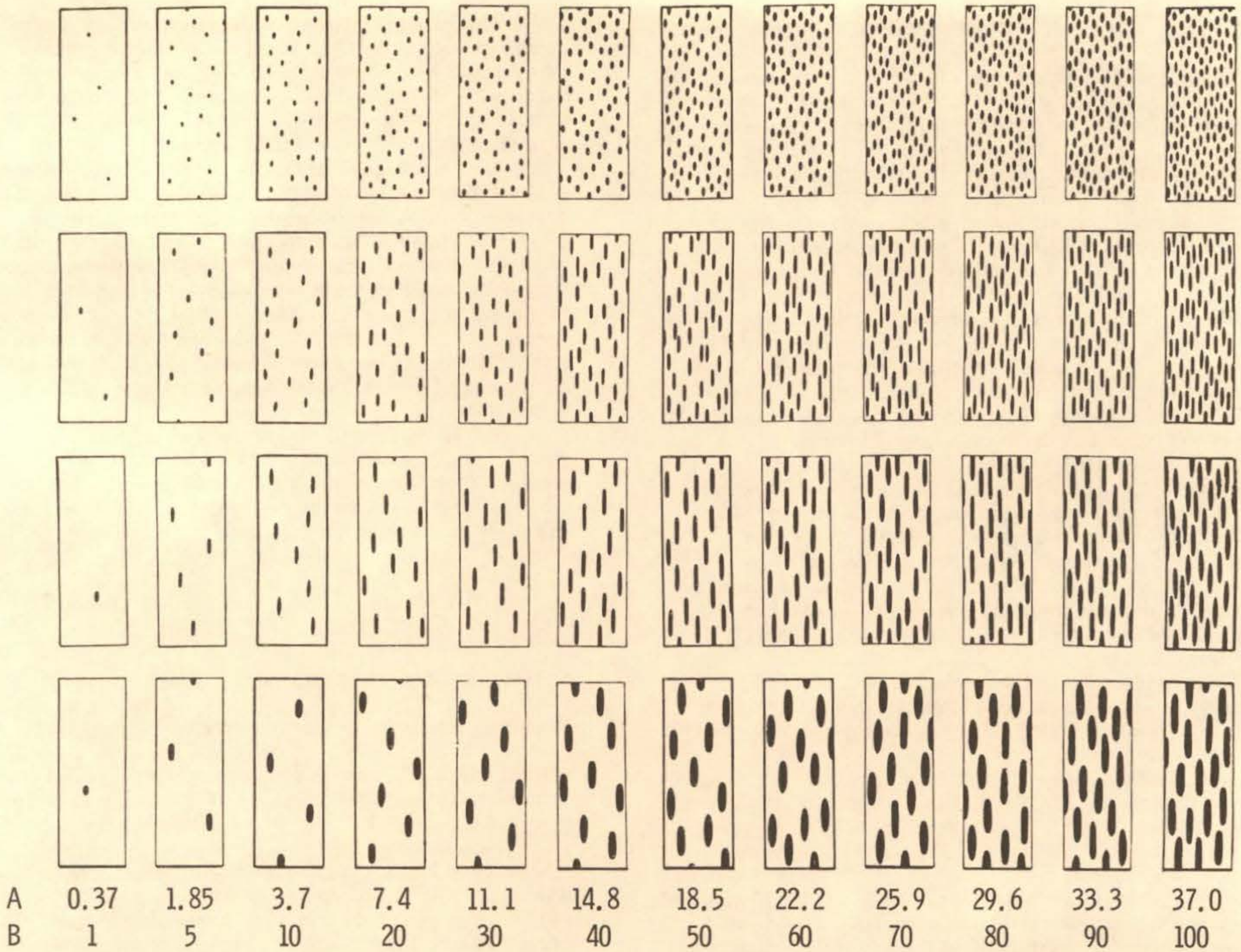


Figure 1. Diagrams to illustrate degrees of rust severity when the uredia are of different sizes (after Peterson, Campbell, and Hannah, 1948). A is the actual percentage of the surface covered by lesions, and B is the visual percentage.

could be measured as precisely from cumulative spore numbers trapped from the air as from the number of uredia on the plants. Thus, a basis was laid for forecasting the severity of rust epidemics. Roelfs, McVey, Long, and Rowell (1972) also correlated rust severity with the cumulative numbers of airborne spores above plots in which the infected wheat grew. Cumulative numbers of airborne spores could be used to measure rust when fields are separated by some distance, but it should not be used in small adjacent plots with different severities of infection where the populations of airborne spores could overlap.

According to Chester (1946) other diagrammatic and descriptive systems to estimate severity of rust were developed by Ericksson and Henning, Butler and Hayman, Yachevski, Nilsson-Ehle, Litvinov, Vavilov, Gassner, Rudorf *et al.*, and Rusakov.

Estimating plant development: Knowledge of the stage of plant maturity when an epidemic begins could help estimate crop loss due to disease. A rust disease that develops on nearly mature cereals causes a smaller loss than it does on younger plants. Feekes (1941) developed a scale, which was modified by several scientists (Large, 1954, and Keller and Baggiolini, 1954), that aided in rapid assessment of cereal plant maturity and provided an accurate comparison of plant development in different locales, seasons, and conditions. We used a scale developed by R.W. Romig (figures 2 and 3) that helped us to easily record more stages of wheat plant maturity throughout the life of the plants than was possible with other scales.

Manipulating rust severity: Some researchers (Doling and Doodson, 1968) studied rust development and yield losses in natural epidemics; but such work is slow because an adequate range of epidemics may not occur annually, the cost of experimentation is high, and comparison is difficult.

Kingsolver and his associates (1959) demonstrated that manipulation of inoculum could help control epidemics. They varied the amounts of inoculum applied to the plots and the distance between inoculum sources and plots and evaluated the effects of different stem rust epidemics on yields of wheat.

Recently Romig and Calpouzos (1970) suggested that stem rust epidemics of different types could be controlled on wheat by varying the time of inoculation and by the judicious application of the fungicides zinc ion-maneb complex (Dithane M45[R]) and nickelous sulfate hexahydrate at rates varying from 1.1 to 2.2 kg/hectare.

Figure 2. The Romig scale for assessment of the growth stages of wheat plants.

Growth Stage	Description
1	One shoot
2	Beginning of tillering
3	Tillers formed, leaves often twisted spirally In some varieties of winter wheats, plants may be 'creeping' or prostrate.
4	Beginning of the erection of the pseudo-stem, leaf-sheaths beginning to lengthen
5	Pseudo-stem (formed by sheaths of leaves) strongly erected
6	First node of stem visible at base of shoot
7	Second node of stem formed, next-to-last leaf just visible
8	Last leaf visible, but still rolled up; head beginning to swell
9	Ligule of last leaf just visible
10	Boot stage, sheath of last leaf completely grown out, head swollen but not yet visible
11	Awns just showing
12	Heading – 1/4 of heading process completed
13	Heading – 1/2 of heading process completed
14	Heading – 3/4 of heading process completed
15	Heading – 95 percent of heading process completed
16	Beginning of flowering
17	Flowering – complete to top of head
18	Flowering – complete to base of head
19	Kernels near middle of head 1/8 formed
20	Kernels near middle of head 1/4 formed
21	Kernels near middle of head 1/2 formed
22	Kernels near middle of head 3/4 formed
23	Kernels fully formed, contents watery
24	Early milk
25	Milk
26	Late milk
27	Early dough
28	Mid-dough – kernel soft but dry
29	Late dough – kernel hard but not ripe
30	Ripe
31	Harvest

Presentation of data: Disease progress curves have been used to define and measure the course of epidemics because they have been the simplest and clearest way to present the data (Van der Plank, 1963, and Zadoks, 1961). These curves are usually sigmoid because in the early stages of the epidemic most spores fell on healthy tissue, while at the latter stages many spores landed on diseased tissue.

For convenient analysis by statistical procedures, the sigmoid curve should be transformed into a straight line by one of several techniques: probits, logits, logs, etc. (Large, 1966). Variations in the slope and origin of the line would indicate the effects of various factors on disease progress. Probits were advocated by Horsfall (1945) and have been a useful tool for some diseases, but they are probably inappropriate for rust studies, according to Van der Plank (1963), because rust is not usually bionomially distributed in a homogenous field. He considered logits, $\log_e [X/(1\text{-}X)]$, a superior transformation and demonstrated that logits plotted against time provided a straight line for many rust epidemics, where X was rust severity and 1-X was the correction factor that accounted for the fact that not all tissue was rusted.

When rust was severe, 1-X did not fully compensate for the increased severity and the line of the disease progress curve tended to flatten or drop down.

MEASURING YIELD AND LOSSES

One of the most important effects of crop disease is yield loss. We now review concepts, approaches, and interpretations that are related to the measurement of yield.

Yield loss may be related to disease in simple terms as expressed by the following equation:

$$\text{Loss} = Y_h - Y_d$$

where Y_h is the yield of the healthy plants and Y_d is the yield of the diseased plants. The accuracy of the loss estimate will depend on the accuracy of yield measurements.

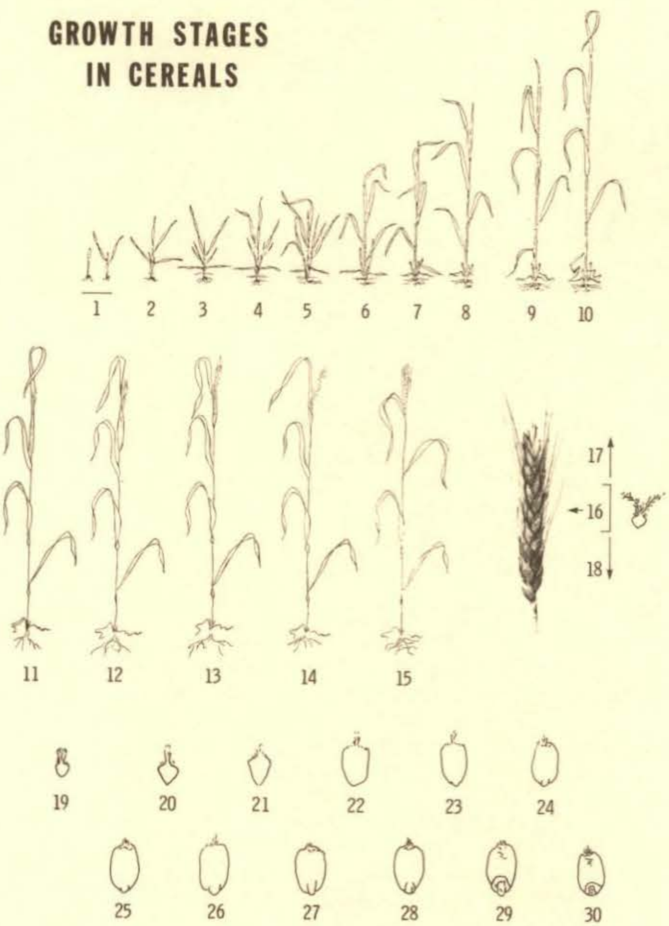


Figure 3. Diagrams representing the growth stages used in the Romig scale (figure 2) to assess the growth of wheat.

The word "yield" involves one or more quantitative or qualitative, sometimes diverse, plant characteristics:

size of plant part (e.g., most fruits and vegetables),
number of a plant part (number of ears on a corn plant),
morphology of a plant part (shape of a potato, for grade quality),
exudate from a plant (latex from a rubber tree),
extract from a plant (essential oil from peppermint),
color (ornamentals, tomatoes),
taste (all food of plant origin),
odor (melons, many other fruits),
texture (crispness of many fruits and vegetables), and
gaseous emissions (carbon dioxide or oxygen yields from plant communities).

"Loss" implies that a reference point exists. The reference point in the equation given above is Y_h. Broadly speaking, two kinds of Y_h are possible, the maximum yield (Y_h max.) and the normal yield (Y_h norm.). Plant breeders and physiologists who study the ultimate genetic capacity of plants for production could use the maximum yield reference point (Y_h max.). Y_h max. can increase quantitatively as new and improved genotypes are developed and as environment is manipulated to maximize crop yield. This concept has been developed primarily under experimental or nonconventional conditions such as growth chambers with enriched carbon dioxide atmospheres, but it might be relevant to conventional techniques of crop management. For our purposes the usefulness of Y_h max. was limited, because the relationship of Y_h max. to Y_d could have been different from that of an open field environment involving Y_h norm.

The value of Y_h norm. is derived under prevailing ecological conditions. However, normal environments are "imperfect" and the yield of a crop may not equal its genetic potential. Y_h norm. has been derived in several ways. Sometimes yields have been averaged on one variety growing disease-free in one region for several years. Sometimes yields from disease-free fields in widely scattered areas in one season have been averaged. Neither of these methods necessarily provides an accurate value for Y_h norm. because environmental conditions can vary annually in one area or among localities in one season. According to one unpublished example, the average yield of a given variety was approximately 30 bushels per acre for several disease-free years. The next year the crop was infected with moderate leaf rust, but its yield was several bushels per acre greater than the previous average. In the disease-free years, moisture levels had been suboptimal and the plants had not produced their top yields. The low moisture, however, also had inhibited development of the disease. In the next year growing conditions were good for both the host and the pathogen. Had the pathogen been controlled, the Y_h norm. probably would have been higher than the recorded yield but no scientific proof supports the probability.

The Y_h norm. and Y_d can be determined by several factorial experiments with different strengths and weaknesses. Generally in factorial experiments, a group of diseased plants is grown next to a group of disease-free plants under the same environmental conditions. Fungicides can be used to prevent the disease from spreading to the Y_h norm. plot. However, fungicides might not be effective for the following reasons:

1. The chemical might only partially control the disease.
2. The chemical might affect yield independently of disease control by acting as a nutrient in the plant (increased yield) or by direct phytotoxicity (reduced yield).
3. The chemical might increase yield by controlling other diseases or pests in addition to the disease under study.

Isogenic lines also can prevent the spread of disease. Isogenic lines theoretically are exactly alike in all respects affecting yield except for resistance to disease. However, the number of available isogenic lines of a crop is limited, the lines are laborious to develop, the lines available might not meet the particular research needs, and the isogeneity of the lines could be questionable.

In other experiments leaves were clipped to simulate the effect of a defoliating disease on yield (Chester, 1946). If only the foliage is diseased and there is no other effect than to reduce the photosynthetic area, mechanical clipping could work, but leaf pathogens often have other effects. Pathogens can secrete toxins that depress general plant vigor or alter the flow of metabolites within the plant (Wood, 1967). Mechanical removal of roots could create similar disparity to simulate damage by root pathogens. Results of removal of plant parts should be compared carefully with the actual effects of disease before clipping is used as a tool for determining Y_h norm. and Y_d.

Where possible, scientists should use several methods for establishing Y norm. and Y . The accuracy of yield measurement is affected by plot size and shape (James and Shih, 1973). They experimented with different lengths of harvested rows and measured the coefficient of variance of yield within replications. The variance in yield among plots treated equally decreased rapidly as plot size increased from less than 9 m to about 27.5 m of row. They recommended that the minimum plot size should include about 46-55 m of harvested rows, and larger plots should be used where possible. The authors concluded that a 5-meter row is too small for accurate measurement of yield when plants are harvested by conventional plot machinery.

Most reports describe a decrease in variance of yield from long, narrow experimental plots (e.g., Wiebe, 1935). However, other reports show that either there is no difference due to plot shape or yield variation is smaller among nearly square plots than among long, narrow ones (Thompson, 1934, and James and Shih, 1973). The available data have not indicated an ideal plot shape, and plot size seems to influence the precision of yield measurement more than plot shape does.

The yield of a given crop in a given field has several facets (figure 4): the part the farmer harvests, the portion he leaves in the field because of inefficient harvest techniques, the portion removed from the plant by physical (e.g., wind and water) and biotic (e.g., birds, insects, mammals) agents. Calpouzos *et al.* (1971) measured yield losses caused by a mechanical plot harvesting method. The experiment and results are described in later sections of this bulletin. The main point is that the accuracy of disease/yield studies can be increased if the components of yield loss due to causes other than disease can be controlled. We suggest that a more accurate estimate of grain loss can be obtained by a "micro sample" method in which each spike in a 3-m row is harvested carefully by hand rather than by the normal method using mechanical cutters and plot threshers. This suggestion may contradict the earlier statement that 5-m of harvested row are not enough to measure yield accurately. However, the "micro sample" method probably avoids losses that confound the results from mechanical harvest methods in small plots. Study of the dynamics of yield development could lead to improved methodology for estimating crop losses due to disease. Conventionally, yield is represented as a static rather than a dynamic process. Usually yield is measured only at harvest. This static concept could explain inadequately the effects of disease on yield. However, study of the harvestable portion of the plant from the time it begins forming until maturity, both with and without disease, could provide more complete information on how disease affects yield. Some interesting possibilities of such a study will be illustrated by a preliminary series of experiments described later in this bulletin.

The authors hope that some of the speculations, suggestions, and questions raised here about the concept of yield may prove helpful to those researchers who are involved fundamentally with the procedure for measuring yield.

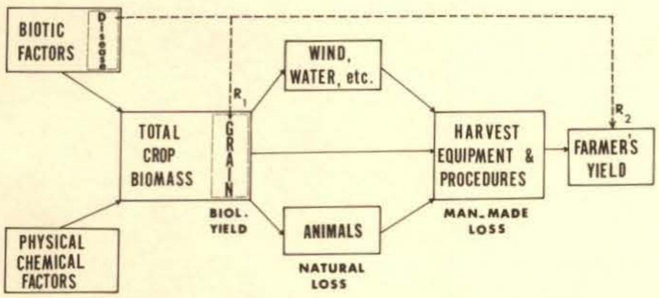


Figure 4. **A diagram of the types of cereal crop yields and loss. R_1 indicates the close relationship between plant disease and biological yield; R_2 indicates the more remote relationship between plant disease and the farmer's yield.**

METHODS AND MODELS FOR PREDICTING LOSSES CAUSED BY THE CEREAL RUSTS

Crop losses caused by disease have been noted throughout history. The first modern published method to estimate losses caused by wheat stem rust was by Kirby and Archer (1927), who developed a table (table 1) to standardize the estimation of loss. If the time intervals between wheat growth stages were equal, a constant rate of loss from disease and increasing disease severity would be expected. Experience has shown that estimates of loss from the table are improved if more than one observation can be made.

Table 1. Table for computing the percentage of loss of yield of wheat because of stem rust (from Kirby and Archer, 1927)

Disease severity percentage at different crop stages

Boot	Flower	Milk	Early dough	Late dough	Ripe	Loss from stem rust (%)
–	–	–	–	Tr	5	0.0
–	–	–	Tr	5	10	0.5
–	–	Tr	5	10	25	5
–	Tr	5	10	25	40	15
Tr*	5	10	25	40	65	50
5	10	25	40	65	100	75
10	25	40	65	100	100	100

*Tr = trace

Before Kirby and Archer, scientists generally estimated losses by questioning people about actual and expected yields, by comparing the present yield with previous yield from the same area, and, with the advent of resistant varieties, by comparing yields of rusted and rust-free crops (Chester, 1950). Therefore, the standardization by Kirby and Archer stimulated further research despite its shortcomings. The major deficiencies in their system are: 1) the disease increase rate is fixed, 2) the relationship of yield loss to disease severity is similar regardless of host genotype, and 3) the effects of similar amounts of disease are equal regardless of location or distribution on the host plant.

The intensive research of Greaney and co-workers in Canada during the late 1920's and early 1930's was the first major effort to use data from experiments in which wheat and oat stem rust had been controlled chemically to estimate losses. This work (Greaney, 1935) is summarized in tables 2 and 3. Those data were based on hundreds of plots ranging from 9.3 to 93 m^2. A straight line regression indicated that the relation was linear between terminal disease and yield. Thus, in Greaney's experiments, each 10 percent increase in rust severity caused an average loss of 5.4 percent (range 3.1-9.7) for wheat stem rust and 5.0 percent (range 2.3-7.0) for oat stem rust.

Table 2. Actual yields of stem rusted and rust-free Marquis wheat at Winnipeg, Manitoba, from 1925 to 1932 and the calculated reduction in yield for each 10 percent of rust severity (from Greaney, 1935)

Year	Yield		Terminal stem rust severity	Yield reduction for each 10% of rust severity	
	Rust-free Bu/A	Rusted Bu/A	%	Bu/A	%
1925	55.0	12.5	85	5.3	9.7
1926	45.0	42.0	15	–	–
1927	48.8	12.2	85	3.6	7.4
1928	35.0	22.0	48	–	–
1929	29.0	24.5	30	2.0	6.9
1930	30.4	6.1	90	2.5	8.2
1931	22.8	9.4	76	1.8	7.9
1932	48.2	31.2	70	1.5	3.1
Mean	39.3	20.0	63	2.1	5.4

Table 3. Actual yields of stem rusted and rust-free Victory Oats at Winnipeg, Manitoba, in 1930, 1931, and 1932 and the calculated reduction in yield for each 10 percent of rust severity (from Greaney, 1935)

Year	Yield		Stem rust severity	Yield reduction for each 10% of rust severity	
	Rust-free Bu/A	Rusted Bu/A	%	Bu/A	%
1930	74.8	29.5	70	4.7	7.0
1931	45.3	23.8	65	2.6	5.8
1932	65.6	56.4	56	1.5	2.3
Mean	61.9	36.6	64	2.9	5.0

Chester (1943) predicted losses caused by wheat leaf rust (*Puccinia recondita* Rob. ex Desm.) in Oklahoma 2 1/2 months before harvest (figure 5). His system was based on the fact that leaf rust overwinters as mycelium in winter wheat and reinfects the wheat during occasional warm periods throughout the winter. Chester found that the amount of rust present on April 1 (May 1 in Illinois and Iowa) appeared to be directly proportional to the amount of loss in crop yield. In Oklahoma, Young and co-workers (personal communication) now make a leaf rust severity survey in early April and then estimate losses from Chester's table (1946) (table 4). Chester's table was developed from experimental data from greenhouse, leaf-clipping, and sulphur-dusting experiments; studies comparing yields between rust-free and rust-epidemic years; and comparisons of yields from rust-susceptible and resistant varieties. Chester observed that the relatively constant increase in rust severity during epidemics should make yield loss estimable after one measurement of disease severity. However, he preferred to appraise rust severity at several stages of rust and crop development. He stated that rust normally should be studied from the heading stage onward except when early severe epidemics make earlier observations desirable.

Stem rust damage to yield of Thorne wheat and Abruzzi rye was studied by Kingsolver *et al.* (1959) who found a close relationship between disease severity and yield loss based on the crop growth stage when rust measured 1 percent (table 5). Previous researchers had used higher rust severity percentages to estimate yield losses. The percentage of yield loss was approximately 50 percent of the terminal disease severity. This loss-terminal disease severity relationship was similar to that found by Greaney (1935). Kingsolver *et al.* did not present data from terminal rust severities of 40 percent or less; however, those percentages are commonly found in the major wheat areas. Kingsolver *et al.* notes that damage to rye from rye stem rust was less than that to wheat from wheat stem rust, even though the severity of the diseases was equal and the plants were in similar growth stages. They attributed the response in rye to the fact that early loss of succulence is normal in rye plants. Loss in rye was 55 percent with 1 percent rust severity at early flowering, but loss in wheat was 80 percent.

Van der Plank (1963) suggested two simple hypotheses to explain the relationship between stem rust severity and yield loss. The first related loss to the severity of disease: doubling the disease severity doubled the loss. The second, "area under the disease progress curve," related loss to time of infection (constant severity), i.e. X amount of rust developing 4 weeks before maturity caused twice the loss that X amount developing 2 weeks before maturity caused. He pointed out that the relationship between yield loss and terminal rust severity depended upon the infection rate: the faster the rate the greater the loss. Furthermore, other variables such as variety tolerance, temperature, and moisture stresses could also affect terminal severity and losses.

Doling and Doodson (1968) predicted that losses from stripe rust (yellow rust) would be equal to 3 times the square root of the disease severity at flowering. They reported a linear relationship between yield loss and disease severity in England. Two formulas for predicting disease loss were developed: Loss (L) = .268 x disease severity (R) + 3.9, and L = 3.01 times $\sqrt{R}$ minus 3.6. Their disease increase rate was averaged from 10 years of tests with spring and winter wheats. For practical purposes a formula relating percentage yield loss to 3 $\sqrt{R}$ was suggested except when head infection was involved.

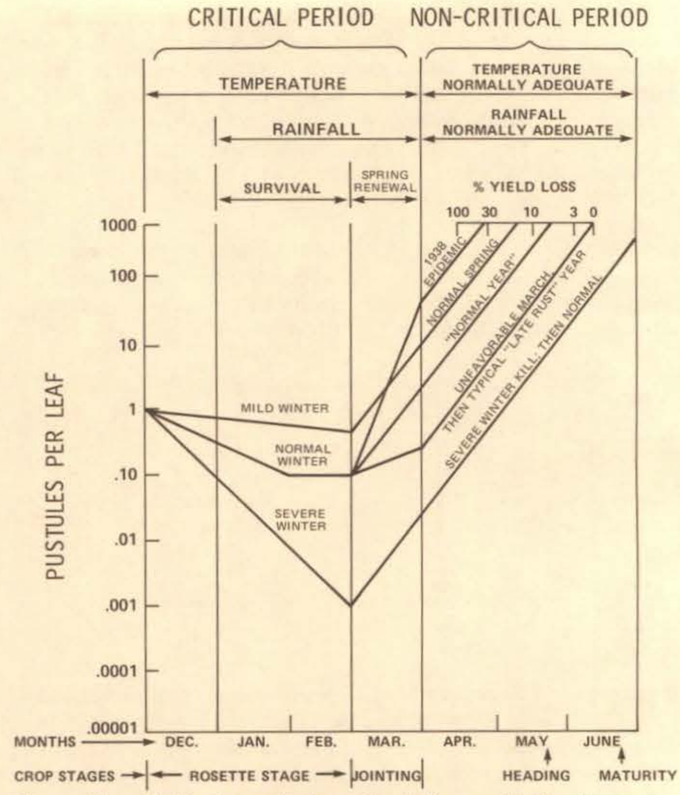


Figure 5. Critical month hypothesis for predicting losses caused by wheat leaf rust (*Puccinia recondita* Rob. ex Desm.) (from Chester, 1943).

They expected that head infection by the disease would cause the yield loss to be underestimated. Mundy (1973) found that 3 $\sqrt{R}$ at flowering underestimated the measured loss. Mundy's data showed L = .442 R + 13.18 or 4.87 $\sqrt{R}$ - 0.13; however, equations based on a later growth stage were L = .44R + 3.15 or L = 5.06 $\sqrt{R}$ - 17.15.

Growing use of protectant fungicides for the control of rust and other foliar disease of wheat in Minnesota and North and South Dakota (Bissonnette *et al.* 1969) required a method to forecast crop losses so that farmers could determine when the sprays are necessary. In South Dakota, Buchenau (1970) devised a scheme to predict loss from wheat stem and leaf rusts (figure 6) based on logarithmic increase in disease per unit of time. This scheme was designed for use with rust severities from .01 pustules per culm to 100 percent infection at 10 days before heading for leaf rust and at heading for stem rust. The scheme was unique at that time because the loss estimate was based largely on an estimated rate of disease increase. The latter was based on meteorological factors. Thus, 100 percent terminal stem rust severity could have a 5, 30, or 57 percent loss associated with it, depending on whether the rate of disease increase was fast, moderate, or slow. Conversely, one stem rust pustule per culm (i.e. 0.1 percent severity) at heading could result in losses of 63, 38, or 11 percent for fast, moderate, and slow rates of disease increase, respectively, depending on weather factors.

Studying four different stem rust epidemics on a single wheat variety at Rosemount, Minnesota, Romig and Calpouzos (1970) and Romig *et al.* (1969) found that loss in plant yield was correlated with disease severity. However, they were

Table 4. Relation between wheat leaf rust severity, wheat growth stage, and yield loss (from Chester, 1946)

Disease severity percentage at different crop stages

Seedling to tillering	Jointing	Boot to heading	Flowering	Milk	Dough	Loss from leaf rust (%)
—	—	Tr	10	25	40	1
—	Tr	10	25	40	65	3
Tr*	10	25	40	65	100	10
10	25	40	65	100	100	20
25	40	65	100	100	100	35
40	65	100	100	100	100	50
65	100	100	100	100	100	70
100	100	100	100	100	100	95

*Tr = trace

Table 5. Loss in yield caused by stem rust associated with a 1 percent severity at various stages of crop growth (from Kingsolver, *et al.*, 1959)

Crop growth stage	Loss from stem rust %
Wheat	
Boot	98
Boot – heading	90
Heading – flowering	82
Flowering – milk	80
Flowering – milk	73
Rye	
Crop growth stage	**Loss from stem rust %**
Heading	71
Early flowering	55
Milk – early dough	40
Milk – early dough	38
Milk – early dough	39

unable to consistently relate loss to the proportional area under the disease progress curve. Their best predictor of loss was the $\log_e$ of disease severity at 3/4 berry stage.

Burleigh *et al.* (1972) studied the relationship between leaf rust severity and crop yield loss using data from 55 winter and spring wheat cultivar-location combinations in a stepwise multiple linear regression program. Coefficients of determination indicated that leaf rust severity at the early dough stage accounted for 64 percent of the variation in yield loss. However, 79 percent of the variation in loss could be explained by combining severity per culm at boot (X_2) with severity per leaf at early berry (X_5) and at early dough (X_7) in a linear regression. Loss was estimated by the equation $Y = 5.3783 + 5.5260X_2 - .3308X_5 + .5019X_7$ with a standard error of 9 percent (table 6). The negative coefficient on X_5 does not seem to have any particular biological meaning.

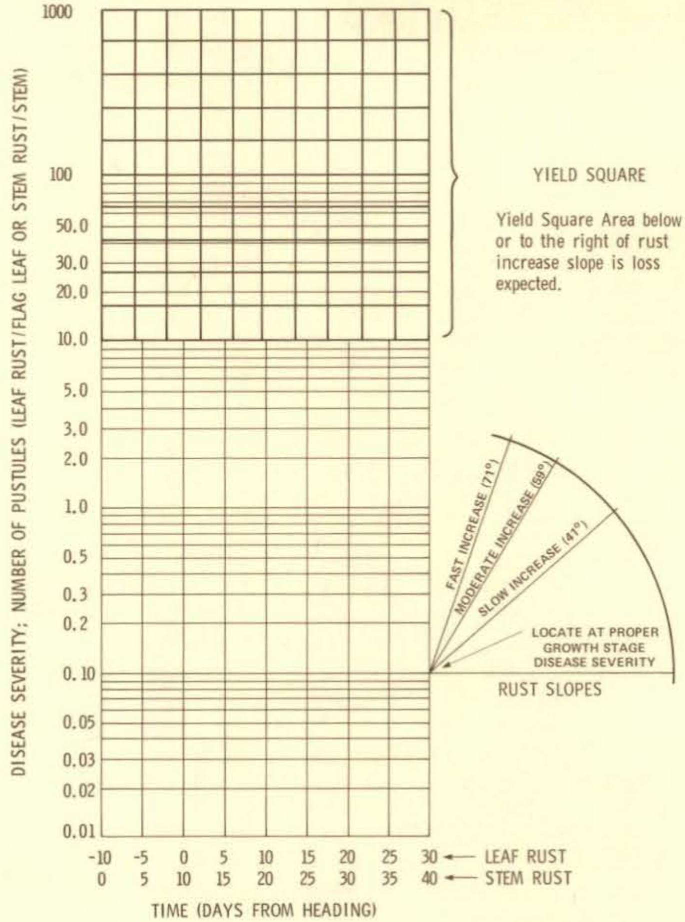


Figure 6. A prediction scheme for losses resulting from wheat leaf and stem rust based on disease severity, stage of crop maturity, and anticipated rate of disease development (from Buchenau, 1970).

Table 6. For selected leaf rust severities, values of the X_2, X_5, and X_7 terms to be substituted into the yield loss formula of Burleigh *et al.* (percent yield loss = $5.3783 + 5.5260X_2 - .3308X_5 + .5019X_7$)

Leaf rust severity %	Postules/ tiller or leaf	$5.5260X_2$ *	$.3308X_5$ †	$.5019X_7$ ‡
.001	1.8/100	0		
.01	1.8/10	0	No loss	
.1	1.8/1	0		
1	18/1	6		
2		11	1	1
3		16	1	2
4		22	1	2
5		28	2	2
10		55	3	5
15		83	5	8
20			7	10
25			3	12
30			10	15
35			11	18
40		100% loss	13	20
45			14	22
50			16	25
60			20	30
70			23	35
80			26	40
90			30	45
100			33	50

* Leaf rust severity per culm at boot stage.
† Leaf rust severity per flag leaf at early berry.
‡ Leaf rust severity per flag leaf at early dough.

MATERIALS AND METHODS
THE MAIN EXPERIMENT

Yield losses were studied during 1969, 1970, and 1971 at Rosemount, Minnesota; Fort Collins, Colorado; and Ponce, Puerto Rico, to provide a wide range of environments in which different types of stem rust epidemics could develop.

Ponce, Puerto Rico (the Fortuna substation of the University of Puerto Rico), is a lowland tropical area within 31 m above sea level at a latitude of 18° N. The soil is an alluvial type. The experiments were made during the winter growing season between November 1970 and April 1971 with temperatures ranging from 18 to 30 C. Dew and rainfall supplemented with irrigation during February and April provided abundant moisture.

Fort Collins, Colorado, is 1,500 m above sea level at latitude 40° N. It is situated on the eastern side of the Rocky Mountains on a high plateau. The temperature during the growing season from early April to July varies from 4.5 to 32 C. Irrigation is necessary because rainfall is slight.

Rosemount, Minnesota, is located on the northern great plains at 45° N at an elevation of 300 m and has an annual rainfall of 62.25 cm. Wheat was planted in late April and harvested in mid August; during that period temperatures varied from 7.5 to 29.0 C.

The wheats studied were of the spring type: Purdue 5481C-1-13-2 (hereinafter called Purdue), a soft red wheat; Mindum (C.I. 5296), a durum wheat; Baart (C.I. 1697), a hard white wheat; and Marquis (C.I. 3641) and Lee (C.I. 12488), hard red wheats. All were susceptible to Race 15B-2 of wheat stem rust but Lee (Hayden, 1956) and Mindum were known to be damaged less by stem rust than the other cultivars.

The wheat was planted early enough at each location for the plants to mature before the end of the growing season. Plots were 4 to 8 rows wide with rows 30.5 cm apart. The experimental design was a random block in which varietal plots for each treatment were adjacent and clustered into a group. The clusters in 1969 and 1970 were separated from each other by 7.6 m of sorghum in Puerto Rico and 7.6 m of barley in Rosemount and Fort Collins. In 1971 the clusters were separated by an unplanted area 3 m wide. The plots were managed to favor plant growth and yield. Plots were replicated from three to five times each year at each location. Data were obtained from each cultivar each year in Minnesota and Colorado, but only from Baart, Lee, and Purdue in Puerto Rico in 1970/71 because Marquis and Mindum did not grow properly in Puerto Rico. During the winter of 1969/70, insect attacks nullified experiments in Puerto Rico. In the winter of 1970/71, the experiments in Puerto Rico provided valuable comparative data because early epidemics developed rapidly and caused high yield loss.

During each year at each location, attempts were made to induce stem rust epidemics with a specific date of disease onset and specific rates of disease increase by inoculation of plants at various dates and by the use of fungicides. The stages of plant maturity selected for the onset of the stem rust epidemic were prior to boot (stage 11), during heading and flowering (stages 12-18), and soon after flowering (stage 19). At each onset stage, we tried to induce epidemics that developed rapidly or slowly. Fungicides were used to slow disease increase and maintain rust-free check plots. We were not able to control, in all plots, epidemic onset and slope according to the preconceived experimental plan. Nevertheless, the data were arranged according to the type of epidemic that occurred.

The fungicides used were nickelous sulfate-hexahydrate and Dithane M-45[R] (zinc ion-maneb complex) both applied at the rate of 1.1 to 2.2 kg of fungicide in 450 to 560 liters of water per hectare. The check plots were sprayed 13-16 times with Dithane M-45[R], or about once a week from the beginning of the tillering stage until the late-dough stage of maturity, and 3-8 times with nickelous sulfate-hexahydrate during the same period, when it appeared that the Dithane M-45[R] treatment was inadequate to prevent disease. Stem rust severity never exceeded 2 percent in the plots that were disease-free checks. Approximately 1 week before the plants reached the stage of maturity that had been designated for the onset of a certain epidemic, the fungicide treatments were withheld and inoculum was applied.

The plots were sprayed during the tiller formation stage of plant development with the fungicide 4-n-butyl-1,2,4-triazole (Indar[R]) produced by Rohm and Haas Co., Philadelphia, Pennsylvania, at rates of 0.56-1.1 kg in 560 liters of water per hectare to prevent leaf rust from developing. This fungicide acts specifically against leaf rust and does not adversely effect stem rust development (von Meyer *et al.*, 1970).

Plots were inoculated with a single isolate of *Puccinia graminis* Pers. f. sp. *tritici* Ericks and E. Henn., race 15B-2 culture number 65-39-2. The purity of the race was checked annually on differential wheat varieties in the greenhouse. Some plots were spray-inoculated with suspensions of uredospores [.01 to 2.0 mg. of uredospores per ml of a nontoxic lightweight mineral oil (Rowell and Hayden, 1956)].

For other plots, water suspensions of uredospores were injected into the leaf whorls of plants on the edges and corners of the plots and the fungus was allowed to spread from the inoculated plants into the plots.

Disease severity and plant growth stages were estimated in each plot many times each growing season: 31 times at intervals of 3 to 7 days in Puerto Rico, 12 to 27 times at intervals of 3 to 8 days in Colorado and 12 to 17 times at intervals of 3 to 6 days in Minnesota. The rust severity scale devised by Peterson, Campbell, and Hannah (1948) (figure 1) was used to estimate disease severity. Rust severity was estimated on the upper and lower halves of the plants at three or four different sites within the plot, then the individual estimates were averaged. The Romig scale was used to estimate wheat growth stages (figures 2 and 3). For each observation several random readings were taken and averaged within each plot.

When the plants were mature, the plots were harvested by two methods. With one method, the heads from 3 m of row were clipped from the plant, air dried at 32 C for 24-36 hours and then hand threshed. With the second method, 16 m of row in the center of each plot was harvested by plot combine or the plants were cut by a machine and then threshed in a Vogel thresher. Care was always taken to harvest only continuous stands in each row. Cleaned seed was weighed and the yield was calculated for each plot. The loss due to stem rust was calculated by the formula,

$$\text{percent loss} = \left[1 - \frac{Y_d}{Y_h \text{ norm}}\right] \times 100.$$

Stem rust severity and plant growth stage were plotted for each observation date for each plot. (See figure 7 for an example.) It was apparent that a straight line could be drawn that would fit most of the data of each epidemic. Standard linear regression was calculated by the formula $\hat{Y} = a + bx$, where a = the origin at the Y axis, b = the rate of increase per unit of growth stage (slope), and x = plant growth stage. The onset of the epidemic was then arbitrarily defined as the point at which the regression line crosses the X axis, regardless of when the first stem rust infection was observed in the plot.

In one of many attempts to relate disease to loss, several epidemics with almost equal slopes, but with onset at different growth stages, were drawn on coordinate paper (figure 8). A vertical scale for yield loss was introduced on the right side of the graph, and the percentage loss was plotted on each epidemic line. When three or more epidemics with similar disease slopes but with differing onset stages were plotted as described, the loss date points showed about a straight line with a negative slope (figure 8). The slope of this yield loss line changed when another group of epidemic disease lines with different slopes was chosen (figure 9). Therefore, it seemed that a single yield loss line could predict loss from all epidemics. We then tried another approach by constructing three-dimensional models that related the rate of epidemic development (slope), the stage of plant growth at the onset of the epidemic, and the percentage of yield loss (figure 10). The models were constructed with pins cut so that their height indicated the percentage yield loss for each experimental plot of each cultivar. The pins were positioned on the grid which represented growth stage by disease slope. Visual inspection of the models showed a regular relationship between yield loss and the other two parameters. A mathematical model was designed from these data and is discussed later.

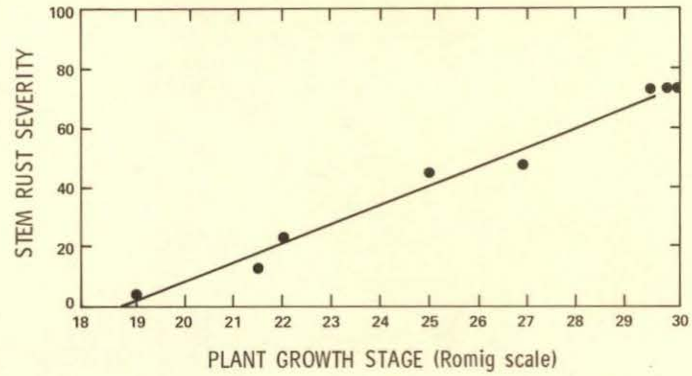


Figure 7. An example of the disease progress observed during the period of this study. This example is for the cultivar Purdue at Rosemount in 1971.

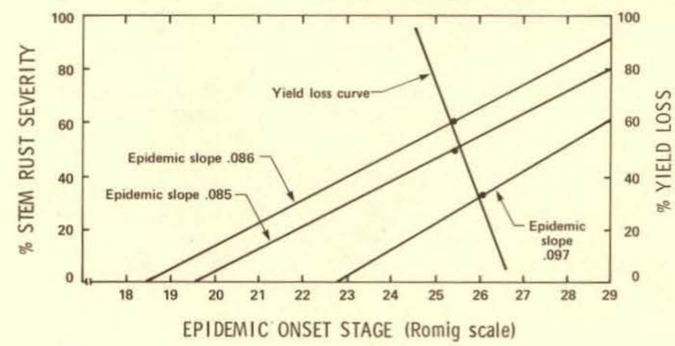


Figure 8. The relationship of yield loss to stem rust epidemics of similar slopes. The yield loss data points fall on or close to a straight line (yield loss line).

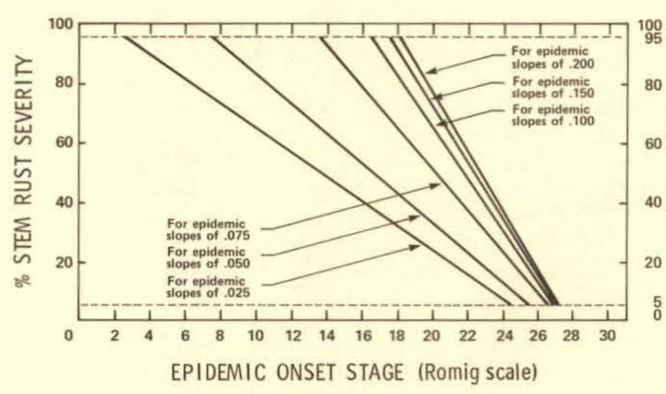


Figure 9. The relationship between yield-loss lines (figure 8) for clusters of epidemics with different epidemic slopes.

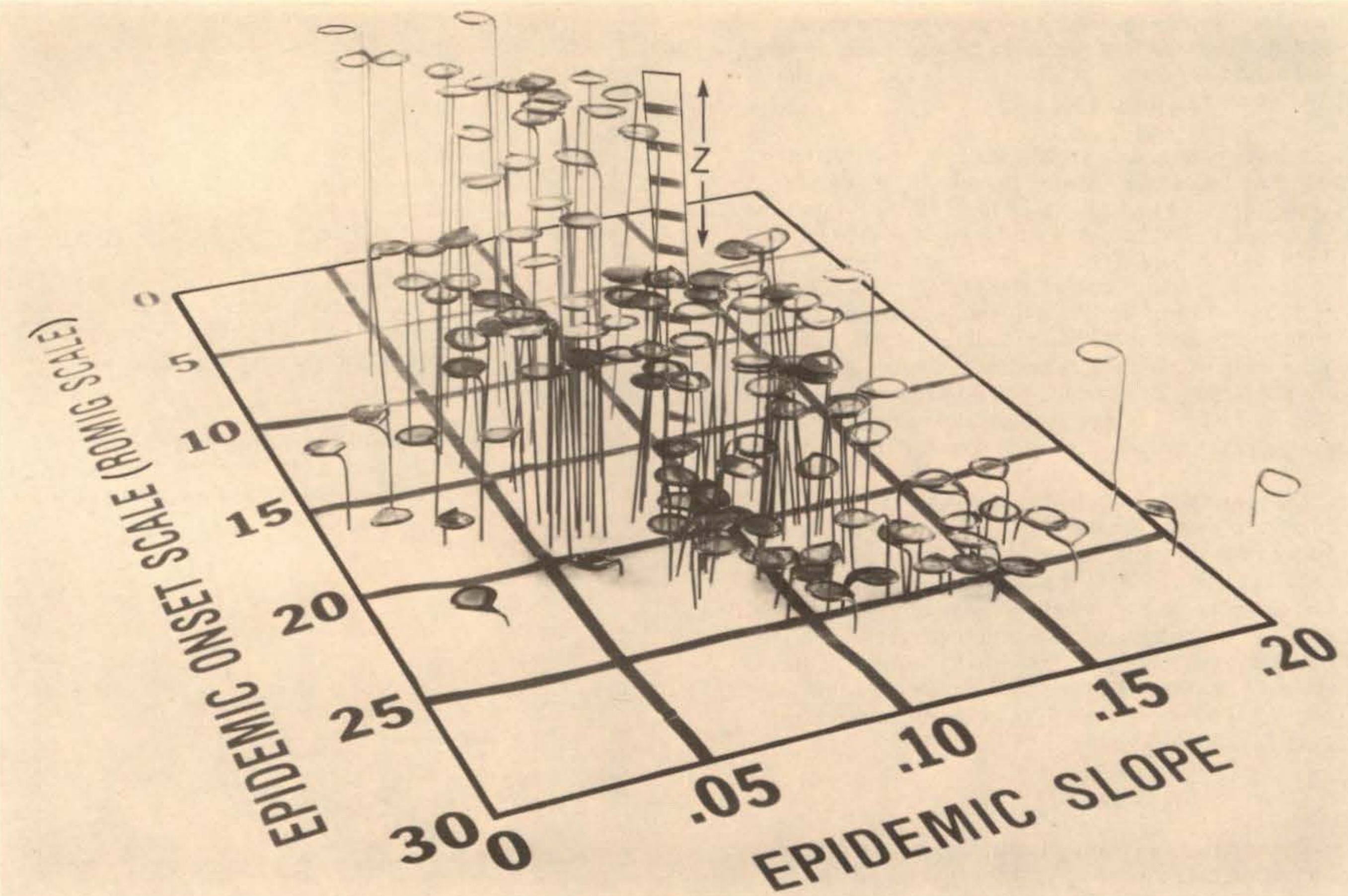


Figure 10. An example of the three-dimensional models that illustrate the relationship between yield losses (Z) and epidemics of stem rust. This model is for the wheat line, Purdue.

THE MECHANICAL HARVEST LOSS EXPERIMENT

Yield loss caused solely by mechanical methods of harvesting plots was measured in a 1970 satellite experiment in Colorado and Minnesota. At each site each of five rust-susceptible wheat cultivars – Baart, Lee, Marquis, Mindum, and Purdue – were planted in plots 2.4 m by 6.1 m. There were five replicate plots for each cultivar and each disease epidemic. At both locations losses were studied in early and late epidemics, as determined by the onset of the epidemics. Early epidemics were those that began at heading stage of plant maturity (stage 15) in Minnesota and at kernel 1/2-formed stage (stage 21) in Colorado. Late epidemics were those that began at kernel 1/2-formed stage of growth (stage 21) in Minnesota and at the milk stage (stage 25) in Colorado. Check plots were kept free of disease with repeated sprays of Dithane M-45R.

At harvest, the rows in each plot were trimmed to 5 m and the outside borders were removed. This gave up to 40 m of row in each plot. Where gaps 30.5 cm long or longer existed within a row, clumps of wheat at each side of the gap were removed to eliminate any border-type effects. The lengths of the solid rows of wheat remaining in each plot were measured. Calculations of yield per unit area were based on actual lengths of rows harvested.

In one of the two different harvest techniques used, three rows each 1 m long were chosen at random from each plot and the wheat heads within each row were plucked and threshed by hand saving every kernel. In the second technique, a total of 15 m was harvested from at least three rows; the plants were cut with a Jari plot cutter and threshed with a Vogel plot thresher. The grain from both harvest methods was uniformly dried for 2 days in a forced-air oven at approximately 40 C, then cooled and weighed.

THE CARYOPSIS EXPERIMENT

In addition to the main field experiment and the one described above, the affect of disease on the dynamic development of yield was studied. Caryopsis dry weight from the time of flowering until ripe stage was measured on rusted and healthy plants during 1969, 1970, and 1971 at the Minnesota site on the cultivars Baart, Era (C.I. 13986), and Marquis.

Plants were sampled from flowering (stage 16) until harvest (stage 31). From each plot, three of the most mature heads were cut by hand from plants from interior rows to avoid border effects on the samples. On Monday and Thursday of each week, plants from plots with rust and without rust (Dithane M-45R-sprayed plots) were sampled. All heads sampled were cut from the same part of the same row. The freshly cut heads were tagged and placed in a dry-ice container until they were brought to the laboratory where they were oven-dried on an open rack at 103 C for at least 16 hours. The heads were then removed from the oven, and the center five caryopses (most mature) were removed from each of the three heads. The 15 caryopses were then weighed to the nearest 0.1 mg. This size of the sample seemed adequate because the error mean square was similar for larger samples (45 caryopses per row).

RESULTS
THE MAIN EXPERIMENT

This section presents data on the types of stem rust epidemics studied and the losses they caused. The data are summarized in appendix table 1 where the epidemics and their losses are grouped according to cultivar, location, year, and onset stage.

Some 469 epidemics of stem rust of many different types were studied. The timely applications of inoculum and fungicides to plots located in different environments during a 3-year period enabled us to study yield loss from a wide range of stem rust epidemics. Table 7 shows the number of epidemics and the average percentage of yield loss in Minnesota, Colorado, and Puerto Rico for each year.

Table 7. Number of stem rust epidemics and associated yield loss. The epidemics are characterized by different slopes and onset stages. Data are for locations and years.*

	Rate of epidemic development (slope)							
	Slow <0.05		Moderate 0.05-0.105		Fast 0.106-0.199		Very fast 0.20-0.30	
Onset stage†	No. of epidemics	% loss	No. of epidemics	% loss	No. of epidemics	% loss	No. of epidemics	% loss
			Minnesota, 1969					
<11	0	–	0	–	0	–	0	–
11-19	2	11	15	48	1	70	0	–
20-30	2	27	13	23	27	31	1	18
			Minnesota, 1970					
<11	2	55	0	–	0	–	0	–
11-19	15	43	37	64	2	68	0	–
20-30	0	–	26	33	19	44	0	–
			Minnesota, 1971					
<11	0	–	0	–	0	–	0	–
11-19	1	9	5	60	0	–	0	0
20-30	0	–	9	23	19	27	6	19
			Colorado, 1969					
<11	0	–	0	–	0	–	0	–
11-19	1	57	0	–	0	–	0	–
20-30	5	25	32	36	18	34	5	35
			Colorado, 1970					
<11	0	–	0	–	0	–	0	–
11-19	0	–	6	64	0	–	0	–
20-30	1	3	19	33	64	27	11	28
			Colorado, 1971					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	14	8	8	9	5	12	6	17
			Puerto Rico, 1970					
<11	6	86	12	96	6	95	5	97
11-19	13	18	15	85	0	–	0	–
20-30	12	11	1	54	2	61	0	–

*Data are totals of epidemics and average losses (rounded off) for the wheats Baart, Lee, Marquis, Mindum, and Purdue in Minnesota and Colorado each year; they are for Baart, Lee, and Purdue in Puerto Rico in 1970.

†Onset stage based on the Romig scale (figures 2 and 3).

In Minnesota the calculated onset stage for most of the epidemics was the early heading stage (stage 11). During the 3 years of the study, onset stage preceded the boot stage in only two epidemics. In Minnesota 31 of the epidemics had slow increase rates (slope <0.05), 105 moderate (slopes between 0.05 and 0.105), 57 fast (slopes between 0.106 and 0.199), and 7 very fast (slopes 0.20 and greater).

In Colorado all of the epidemics had calculated onset stages after the boot stage of plant development (stage 10). Eight of the epidemics had onset stages during the host flowering period (stages 11-19), and 183 had onset stages as the kernels were forming and filling (stages 20-30). In Colorado 21 of the epidemics had slow increase rates, 62 moderate, 89 fast, and 19 very fast.

In Puerto Rico 28 of the epidemics had onset stages preceding the boot stage of host growth, 29 began as the plants were heading and flowering, and 15 began while the kernels were forming and filling. Thirty-one of the epidemics had slow increase rates, 29 moderate, 8 fast, and 4 very fast. Significantly for our study the latter four epidemics in Puerto Rico were unique because they began at stage 11 or earlier and developed swiftly (table 7).

Table 8 shows the number of epidemics on each cultivar during our tests. Fewer epidemics were recorded on Mindum and Marquis because they were not tested in Puerto Rico. The number of epidemics per year is shown in table 9 and was largest in 1970; the range and type were also greatest that year because of the Puerto Rico tests.

Table 8. Number of stem rust epidemics characterized by different onset stage and slopes. Data are for wheat cultivars.*

	Rate of epidemic development (slope)			
Onset stage†	Slow <0.05	Moderate 0.05-0.105	Fast 0.106-0.199	Very Fast 0.20-0.30
		Baart (106 epidemics)		
<11	0	4	0	0
11-19	2	25	1	0
20-30	12	16	32	14
		Lee (101 epidemics)		
<11	4	5	4	1
11-19	6	7	1	0
20-30	10	22	37	5
		Marquis (77 epidemics)		
<11	0	0	. 0	0
11-19	1	11	0	0
20-30	4	22	37	4
		Mindum (77 epidemics)		
<11	2	0	0	0
11-19	13	9	0	0
20-30	6	23	21	3
		Purdue (102 epidemics)		
<11	2	3	2	4
11-19	9	26	1	0
20-30	3	25	27	3

*Data for each cultivar are totals for 1969, 1970, and 1971 from Minnesota and Colorado. In 1970 totals include Puerto Rico data for Baart, Lee, and Purdue.

†Onset stage is based on the Romig scale (figures 2 and 3).

Table 9. Number of stem rust epidemics characterized by different onset stages and slopes. Data are for years.*

	Rate of epidemic development (slope)			
Onset stage†	Slow <0.05	Moderate 0.05-0.105	Fast 0.106-0.199	Very Fast 0.20-0.30
		1969 (122 epidemics)		
<11	0	0	0	0
11-19	3	15	1	0
20-30	7	45	45	6
		1970 (274 epidemics)		
<11	8	12	6	5
11-19	28	58	2	0
20-30	13	46	85	11
		1971 (73 epidemics)		
<11	0	0	0	0
11-19	0	5	0	0
20-30	15	17	24	12

*Data are totals for Baart, Marquis, Mindum, Lee, and Purdue in Minnesota and Colorado. In 1970, totals include Puerto Rico data for Baart, Lee, and Purdue.

†Onset stage is based on the Romig scale (figures 2 and 3).

THE MECHANICAL HARVEST LOSS EXPERIMENT

The disease reached an average maximum severity of about 90 percent and 70 percent in Minnesota and 65 percent and 45 percent in Colorado for the early and late epidemics, respectively. Check plots had at most only traces of disease.

From all cultivars the average yield was less from machine harvest than from hand harvest as follows:

	Minnesota	Colorado
Early epidemic plots	43% less yield	24% less yield
Late epidemic plots	20% less yield	18% less yield
Check plots, disease free	12% less yield	14% less yield

The losses indicated by these yields were attributed directly to the inefficiency of the mechanical harvest procedures and could confound any study of the direct effect of disease on yield loss. (See R_2 in figure 4.) Our data from hand harvest probably relate closely to biological yield (R_1 in figure 4).

The data in table 10 show that the percentage difference between hand and machine harvest was not consistent among cultivars. The difference tended to increase from disease free to late epidemic (moderate disease loss) to early epidemic (more severe disease loss), but there were exceptions to this trend (e.g. Baart in Minnesota).

The yields were always higher from hand than from machine harvest. The calculated average yields expressed as metric tons per hectare (5 cultivars combined) for the check plots were within normal range: in Minnesota 2.72 (machine) and 3.13 (hand), in Colorado 3.40 (machine) and 4.01 (hand). As expected, the actual yields decrease from disease-free to late epidemic to early epidemic plots.

The results of this experiment indicate that, even on disease-free wheat, yield is lower when crops are harvested with conventional machinery (13 percent loss of yield) than when they are hand harvested. Furthermore, the difference in yield between hand and machine harvested crops usually increases when damage from stem rust increases.

Table 10. Yield loss caused by machine harvest as compared to hand picked heads.*

	Minnesota (Colorado)		
Wheat cultivar	Early severe epidemic†	Late severe epidemic	Disease-free check
	%	%	%
Purdue	45 (24)	28 (18)	6 (24)
Mindum	52 (22)	24 (21)	19 (14)
Marquis	32 (17)	19 (17)	8 (8)
Baart	40 (36)	15 (16)	18 (9)
Lee	45 (23)	14 (16)	7 (15)

*[1– (Yield from machine harvest ÷ yield from hand harvest)] x 100.
†Each datum is the average loss for five sets of replicate plots. Yields were adjusted to a comparable unit of harvested area.

THE CARYOPSIS EXPERIMENT

Figures 11 and 12 show the dry weight gains of the wheat caryopses from plants, with and without stem rust. Era and Marquis are hard red spring wheats, and Baart is a hard white spring wheat; Era is resistant to the stem rust race 15B-2 and was included as a disease-free check unsprayed with fungicides. The hard white healthy wheat produced more than the two hard red healthy wheats, and the yields of the latter two were similar, suggesting that the fungicide itself did not affect directly caryopsis weight.

Figure 11 shows the gains in dry weight of caryopses from diseased and healthy plants. After flowering began, all the dry weight curves were similar for 10-14 days, but then the caryopses from diseased plants stopped accumulating dry matter. Stem rust development on Baart was 4 percent severity on day 49 (heading stage) and 86 percent on day 70 (mid-dough stage). On Marquis stem rust development was 4 percent severity on day 54 (flowering completed) and 83 percent on day 70 (early dough). The sudden separation of the healthy and diseased curves is striking. This pattern was noted throughout the study and appears again in figure 12 in which the caryopsis dry weight on the rusted plants tended to increase and decrease in a cyclical manner. (Also see figure 11.) This cyclical pattern could be an artifact due to technique, but the pattern may be real because it is either absent or much reduced in the healthy plants. We have no explanation for the sudden divergence of the healthy and diseased caryopsis development curves or for the cyclical pattern in dry weight of the caryopses on diseased plants.

The results of this preliminary study suggest that the development of caryopses on the plant may provide valuable clues to the fundamental understanding of how disease affects yield.

THE MODEL FOR YIELD LOSS

The extensive data of this study presented the opportunity for examination of a suitable model for yield loss as a function of two rust epidemic parameters, the morphological stage of plant development at the point of epidemic onset and the rate of epidemic development (slope). The stage of onset was defined as the point at which the calculated slope line of the disease increase curve intercepted the plant development axis.

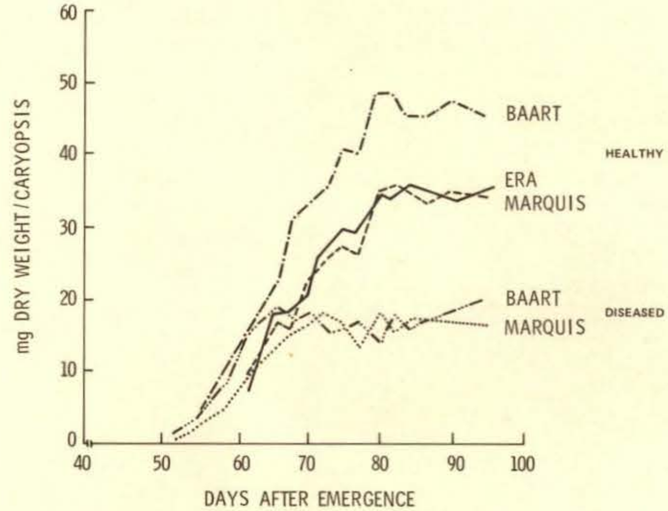


Figure 11. Dry weight gain for caryopses from two wheat cultivars susceptible to stem-rust and one resistant to it (Era). 1970 data.

Figure 12. Dry weight gain for caryopses from Baart wheat with and without stem rust. 1971 data.

The slope line is a least squares fit of field observations on disease severity estimates during the linear expansion phase of the epidemic.

As already described, the data for each of the five varieties were plotted as a three-dimensional model whose typical form is shown in figure 10. Despite the variation inherent in methods of harvest and in the definition of the loss calculation, a response surface was clearly visible in the model for each cultivar.

The extensive data available enable observation of a remarkably strong linear relation between percent of loss and the stage of rust onset at each separate value on the epidemic slope. The actual shape of the relationship changes regularly as a function of the specific epidemic slope. For most slopes between .00 and .20, an onset stage occurs before which losses will be at least 95 percent, and a later onset stage occurs after which losses will not exceed 5 percent.

Losses were truncated so that those greater than 95 percent equaled 95 percent and losses less than 5 percent equaled 5 percent. We assumed no useful information was lost, because minute losses were almost meaningless because of the variation in yield determination and differences in losses close to 100 percent were of no practical meaning.

Truncation of the loss measurements sharpened the upper and lower corners of an otherwise sigmoid relation between percentage yield loss and growth stage at disease onset. This relation was described by a straight line between losses of 5 and 95 percent (figure 13). As the epidemic slope, b, increased from .00 to .20, the line in figure 13 becomes steeper and moves to the right. An upper limit of approximately .20 on the range of slope values studied was adopted. The sparse data beyond this limit would not improve the model. The juxtaposition of the yield loss slopes (figure 9) over the range of possible epidemic slopes forms a response surface of yield loss to the two parameters of epidemic slope and onset stage (figure 14).

The locations of the upper and lower inflection points in figure 14 move in a regular fashion across the grid tracing out the 95 and 5 percent yield loss contour lines. The three-dimensional model, when viewed from above, is a plane figure (figure 15). The epidemics in the upper right shaded area will result in essentially total yield loss, and epidemics in the lower shaded area will cause little or no loss. Any other yield loss contour between 5 and 95 percent can be interpolated on the Y axis as illustrated in figure 15 where the 50 percent contour is midway between the 5 and 95 percent contours. When the 95 percent contour crosses the zero onset boundary, the 50 percent contour can be located by extending the 95 percent contour and proceeding with the described interpolations.

The physical models suggested, as did figure 14, that the 95 and 5 percent contours are, respectively, parabolic and hyperbolic functions. Investigations of a broader family of curves, including the exponential functions, did not improve fit or interpretation of the model. Algebraic expressions for the 95 and 5 percent loss contours were the essential components of a mathematical model for yield loss. Let X_1 denote values on the onset coordinate axis and X_2 denote values on the slope coordinate axis. The parabolic contour (95 percent loss) is described by the locus of points satisfying the functional relationship $X_1 = f_{95}(X_2)$. Similarly, the hyperbolic contour (5 percent loss) is determined by $X_1 = f_5(X_2)$ where, respectively,

$$f_{95}(X_2) = A_1 X_2{}^2 + A_2 X_2 + A_3 \qquad \text{equation (1)}$$

$$f_5(X_2) = A_4(X_2)^{-1} + A_5 \qquad \text{equation (2)}$$

Table 11 has the fitted values of the A_{1-5} parameters for the cultivars investigated.

The algebraic description of percentage loss (denoted by Y) can be derived from the description of the contour surface. The model for percentage yield loss as a function of slope and onset is given by,

$$Y = f(X_1, X_2) \text{ where} \qquad \text{equation (3)}$$

$$f(X_1, X_2) = .95 \qquad \text{when } X_1 \leq f_{95}(X_2)$$

$$f(X_1, X_2) =$$

$$.05 + .90\,\frac{f_5(X_2) - X_1}{f_5(X_2) - f_{95}(X_2)} \qquad \text{when } f_{95}(X_2) \leq X_1 \leq f_5(X_2)$$

$$f(X_1, X_2) = .05 \qquad \text{when } X_1 \geq f_5(X_2)$$

Rust tolerance is measured by the amount by which yield of one cultivar exceeds yield of another cultivar of similar potential yield when both are equally diseased. The location of the 50 percent loss contour can convey information on the ability of a cultivar to tolerate rust. A variety for which the 50 percent contour line is located to the lower left of figure 15 has less tolerance than a variety for which the 50 percent contour line is located higher and to the right. The total area of the graph (figures 15 or 16) may be divided into the portions above and below the 50 percent contour line. The percentage of area below the line is referred to here as "the tolerance index" (T). The last column of table 11 shows the tolerance indices computed for each of the five varieties. Among those five cultivars, Mindum with T = .48, and Marquis with T = .29 were, respectively, the most and least tolerant. Mindum is known as a tolerant variety compared to the other cultivars tested.

In table 11 the relatively low value of $R^2 = .52$ for Mindum is attributed to the fact that few high losses reduced the range of observed losses. In general, attempts to fit this model to cultivars that are regarded as tolerant will result in low R^2 values.

Figure 16 shows a nonlinear least squares fit of the percentage loss response, Y, to the model in equation 3 for the five cultivars. We shall not discuss here the difficulties of developing an effective algorism or the ad hoc procedures used to achieve convergence. We had modest success and obtained good fits for the proposed model (table 11).

In figures 15 and 16, lack of data could cause the vertex of the $X_1 = f_{95}(X_2)$ parabola to be inside the grid limits. Because it makes no sense for yield loss to recede as epidemic slope increases and onset occurs earlier, the 95 percent loss contour would then be modified to extend horizontally from the vertex to the right boundary of the grid. The paucity of data on high slope epidemics at the late stages of development is responsible for that circumstance.

A deliberate effort to create epidemics that would be distributed widely over the grid was a key feature of this work. Nevertheless, most of the successfully induced epidemics fall in a band that might be described as the area of probable natural epidemics. The few epidemics with high slope and early onset were induced only with artificial inoculation in Puerto Rico. Even so, this band of induced epidemics represents a wider spectrum than could be ordinarily expected and creates a useful foundation for testing the model.

At least 70 observations (table 11) were used to fit the model for each of the five cultivars tested, and the question of the minimal number of observations required to adequately duplicate the yield-loss contour map (figures 15 and 16) for

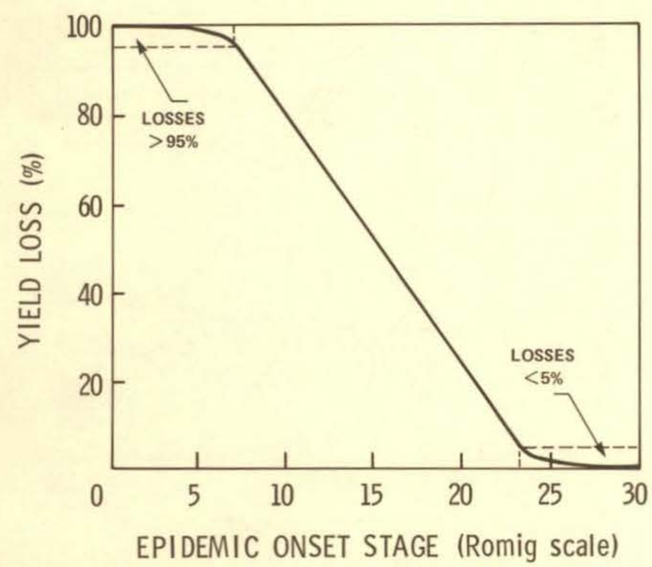


Figure 13. An example of the relation of yield loss to the calculated epidemic onset stage (Romig scale).

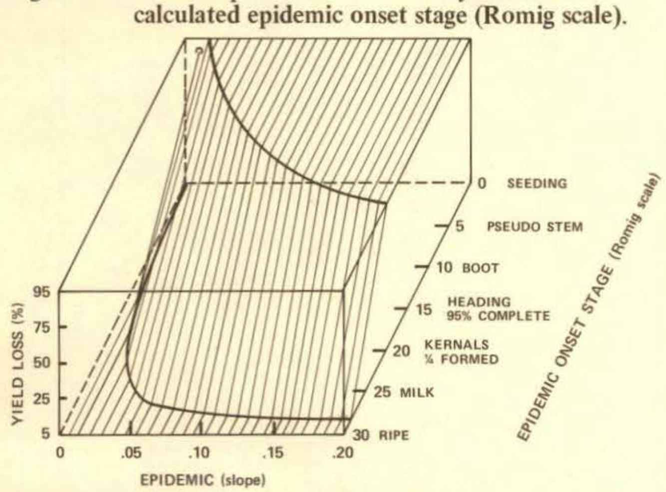


Figure 14. Yield loss as related to epidemic slope and onset stage. The three-dimensional illustration shows the response surface of yield loss to the two parameters of the epidemics.

another untested cultivar becomes a practical issue. A random stratified selection of 12 epidemics consistently produced close approximations to the models exhibited. In nature, however, the desired set of 12 widely dispersed epidemic types might be difficult to establish. A wide distribution of epidemics in the reasonable range is a critical factor.

The generalized model shown in figure 15 was developed by combining the data on the five susceptible cultivars studied. Because of program limitations, a random selection of N = 374 epidemics was used. Our R^2 value of .69 indicates that the generalized model may have sufficient predictive power for practical use in various geographical areas where other susceptible cultivars are prevalent.

These models might be used to predict yield loss but values for the calculated onset of the epidemic and the rate of epidemic development (slope) must be estimated. These values can be obtained in the following manner. The onset of the epidemic is estimated from two sequential observations on stem rust severity made as the epidemic is increasing linearly, i.e. when the rust severities are between 5 and 95 percent.

These two observations are then plotted in figure 17, and a line is drawn to intercept the observations and the X-axis. Thus, the onset of the epidemic is read from the X-axis. The approximate slope of the epidemic is determined by superimposing the information developed in figure 17 onto figure 18. Finally, yield loss is determined by locating the value for epidemic onset (as determined on figure 17) and epidemic slope (as determined on figure 18) on figure 15 (the generalized model) or figure 16 (specific cultivar models). The point where the two values meet indicates the yield loss by linear interpolation between the 95 and 5 percent loss contours. Sometimes an investigator may find a cultivar with disease-yield loss relationships that closely resemble those of one of the specific cultivar-yield-loss contour maps in figure 16, so that the specific rather than the generalized model could be used.

Table 11. Values for fitting the model $Y = f(X_1 X_2)$, equation (3).

Cultivar	No. of epidemics analyzed	Coefficients* A_1	A_2	A_3	A_4	A_5	R^2	T
Baart	94	−.05	1.70	4.60	−1.46	27.45	.81	.41
Lee	96	−.18	4.37	−14.00	−1.13	28.36	.81	.40
Mindum	74	−.45	7.30	−22.39	−29.21	31.00	.52	.48
Marquis	73	−.01	.61	11.21	−.08	26.93	.76	.29
Purdue	89	−.19	4.61	−10.58	−7.14	27.28	.81	.37
The combined set	374	−.19	4.73	−13.39	−.32	28.60	.69	.35

*A_1 to A_3 = coefficients of the 95 percent loss contour. See equation 1.
A_4 to A_5 = coefficients of the 5 percent loss contour. See equation 2.
R^2 = coefficient of determination (percent of variation in losses explainable by the model).
T = tolerance index.

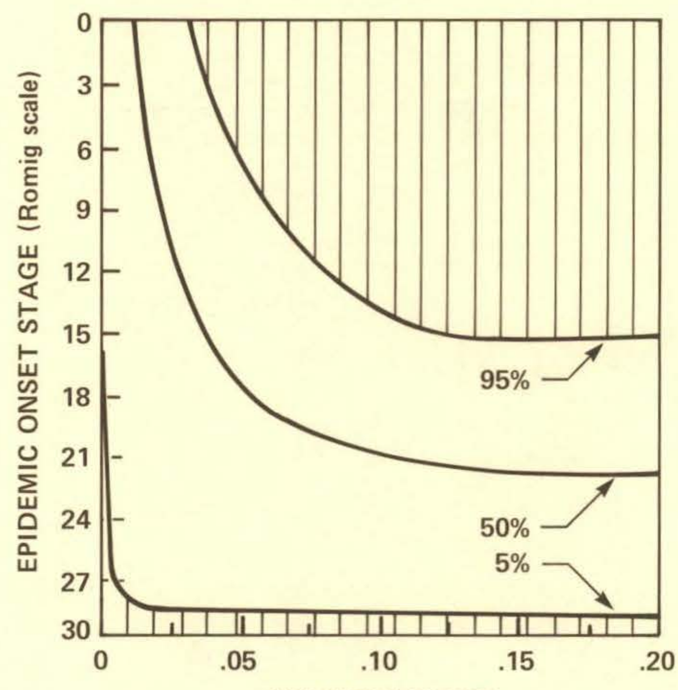
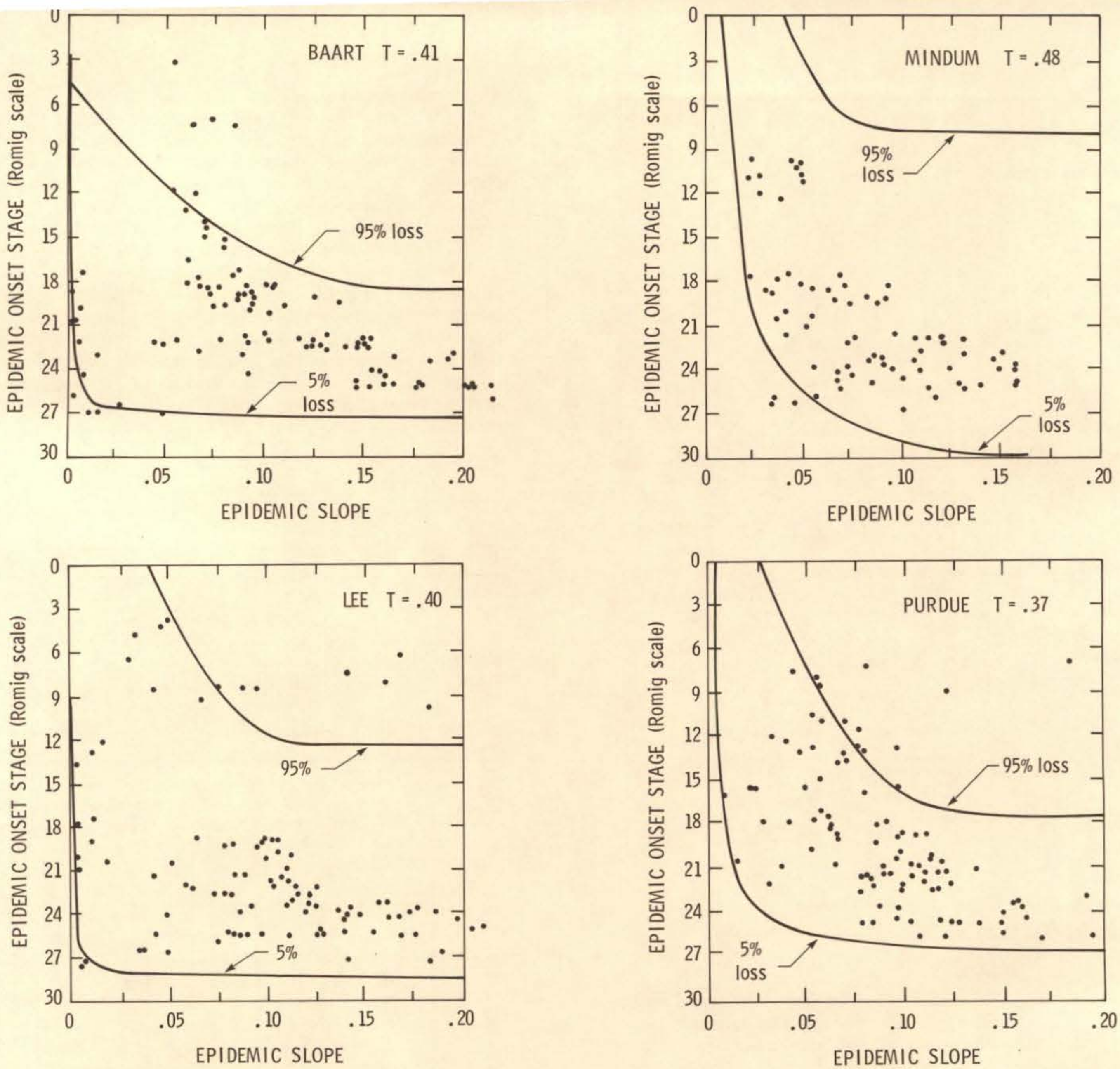


Figure 15. Yield loss contours computed from 374 stem rust epidemics.



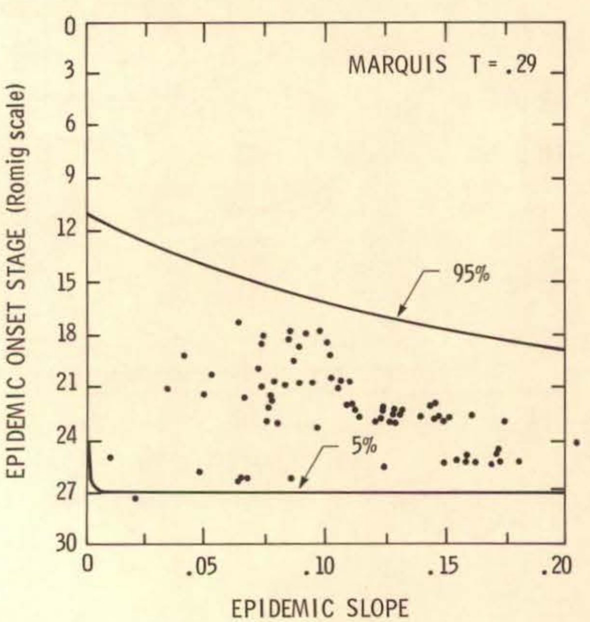


Figure 16. Epidemics of stem rust (black dots) on Baart, Lee, Marquis, Mindum, and Purdue showing the 5 and 95 percent loss contours determined by the statistics in table 11. Tolerance index "T" is shown for each cultivar.

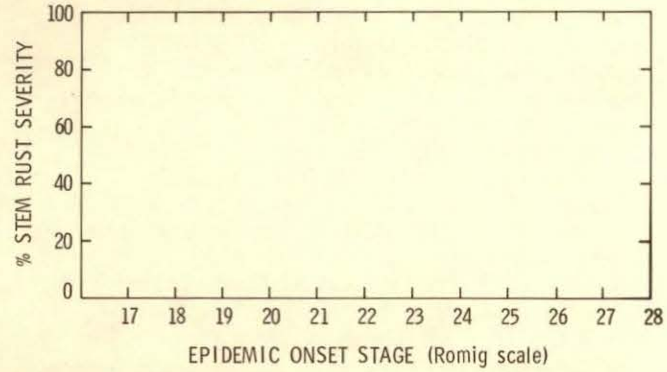


Figure 17. A grid of stem rust severities and epidemic onset stages to be used for estimating the onset of any epidemic.

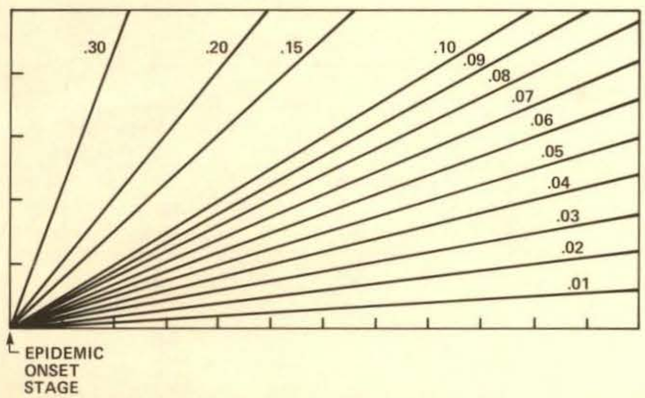


Figure 18. Diagram of stem rust epidemic slopes to be used in estimating slopes of any epidemic.

Literature Cited

Bissonnette, H.L., J.B. Rowell, and L. Calpouzos. 1969. Chemical control of foliar diseases of cereals. Minnesota Sci. 25:8-10.

Buchenau, G.W. 1970. Forecasting profits from spraying for wheat rusts. South Dakota Farm & Home Res. 21:31-34.

Burleigh, J.R., A.P. Roelfs, and M.G. Eversmeyer. 1972. Estimating damage to wheat caused by *Puccinia recondita tritici.* Phytopathology. 62:944-946.

Burleigh, J.R., R.W. Romig, and A.P. Roelfs. 1969. Characterization of wheat rust epidemics by numbers of uredia and numbers of urediospores. Phytopathology 59:1229-1237.

Calpouzos, L., M.E. Madson, and J.R. Welsh. 1971. The effect of harvest technique on recovery of wheat from experimental plots with and without stem rust. Phytopathology (Abstr.) 61:1022.

Chester, K.S. 1943. The decisive influences of late winter weather on wheat leaf rust epiphytotics. Plant Dis. Reptr. Suppl. 143:133-144.

Chester, K.S. 1946. The nature and prevention of the cereal rusts as exemplified in the leaf rust of wheat. Chronica Botanic Co., Waltham, Mass. 269 p.

Chester, K.S. 1950. Plant disease losses; their appraisal and interpretation. Plant Dis. Reptr. Suppl. 193:190-362.

Cobb, N.A. 1892. Contribution to an economic knowledge of the Australian rusts (Uredineae). Agr. Gaz. N.S. Wales. 3:44-68, 181-212.

Doling, D.A., and J.K. Doodson. 1968. The effect of yellow rust on the yield of spring and winter wheat. Br. Mycol. Soc. Trans. 51:427-434.

Feekes, W. 1941. De Tarwe en haar milieu. Vers. XVII Tech. Tarwe Comm. Groningen, 560-561.

Greaney, F.J. 1935. Method of estimating losses from cereal rusts. Proc. World's Grain Exch. & Conf. Regina, Canada. 1933. 2:224-235.

Hayden, E.B. 1956. Progressive development of infection by *Puccinia graminis* var. *tritici* Eriks. and E. Henn. (Guyot) on certain varieties of wheat and the relation of stem rust to yield. Ph.D. thesis. University of Minnesota. 103 p.

Horsfall, J.G. 1945. Fungicides and their action. Chronica Botanica Co. Waltham, Mass. 239 p.

James, W.C., C.S. Shih, W.A. Hodgson, and L.C. Calbeck. 1972. The quantitative relationship between late blight of potato and loss in tuber yield. Phytopathology 62:92-96.

James, W.C., and C.S. Shih. 1973. Size and shape of plots for estimating yield losses from cereal foliage diseases. Exp. Agr. 9:63-71.

James, W.C. 1974. Assessment of plant diseases and losses. Annu. Rev. Phytopathol. 12:27-48.

Keller, C., and M. Baggiolini. 1954. Les stades repères dans la végétation du Blé. Revue Romande, Lausanne. 10:17-20.

Kingsolver, C.H., C.G. Schmitt, C.E. Peet, and K.R. Bromfield. 1959. Epidemiology of stem rust: II (Relation of quantity of inoculum and growth stage of wheat and rye at infection to yield reduction by stem rust.) Plant Dis. Reptr. 43:855-862.

Kirby, R.S., and W.A. Archer. 1927. Diseases of cereal and forage crops in the United States in 1926. Plant Dis. Reptr. Suppl. 53:110-208.

Large, E.C. 1954. Growth stages in cereals. Illustration of the Feekes scale. Plant Pathology 3:128-129.

Large, E.C. 1966. Measuring plant disease. Annu. Rev. Phytopathol. 4:9-28.

Melchers, L.E., and J.H. Parker. 1922. Rust resistance in winter wheat varieties. Bull. U.S. Dept. Agric. 1046.

Mundy, E.J. 1973. The effect of yellow rust and its control on the yield of Joss Cambier Winter Wheat. Plant Pathol. 22:171-176.

Peterson, R.F., A.B. Campbell, and A.E. Hannah. 1948. A diagrammatic scale for estimating rust intensity on leaves and stems of cereals. Can. J. Res. Sect. C. 26:496-500.

Roelfs, A.P., D.V. McVey, D.L. Long, and J.B. Rowell. 1972. Natural rust epidemics in wheat nurseries as affected by inoculum density. Plant Dis. Reptr. 56:410-414.

Romig, R.W., L. Calpouzos, and M.E. Madson. 1969. The effect of stem rust on yield-loss in spring wheat. Phytopathology (Abstr.) 59:1047.

Romig, R.W., and L. Calpouzos. 1970. The relationship between stem rust and loss in yield of spring wheat. Phytopathology 60:1801-1805.

Rowell, J.B., and E.B. Hayden. 1956. Mineral oils as carriers of urediospores of the stem rust fungus for inoculating field-grown wheat. Phytopathology 46:267-268.

Thompson, R.C. 1934. Size, shape, and orientation of plots and numbers of replications required in sweet potato field-plot experiments. J. Agric. Res. 48:379-399.

van der Plank, T.E. 1963. Plant diseases: Epidemics and control. Academic Press, New York, 349 p.

von Meyer, W.C., S.A. Greenfield, and M.C. Seidel. 1970. Wheat leaf rust: Control by 4-N-butyl-1,2,4-triazole, a systemic fungicide. Science 169:997-998.

Wiebe, G.A. 1935. Variation and correlation in grain yield among 1500 wheat nursery plots. J. Agric. Res. 50:331-357.

Wood, R.K.S. 1967. Physiological Plant Pathology. Blackwell Scientific Oxford. 570 p.

Zadoks, J.C. 1961. Yellow rust of wheat. Studies in epidemiology and physiologic specialization. Tijdschdrift over Planteziekten 67:69-256.

Appendix Table 1. Number of stem rust epidemics and associated yield losses. The epidemics are characterized by different slopes and onset stages. Data are for five wheat cultivars in Minnesota, Colorado, and Puerto Rico during 1969, 1970 and 1971.

	Rate of epidemic development (slope)							
	Slow <0.05		Moderate 0.05-0.105		Fast 0.106-0.199		Very fast 0.20-0.30	
Onset stage*	No. of epidemics	Mean % loss	No. of epidemics	Mean % loss	No. of epidemics	Mean % loss	No. of epidemics	Mean % loss
			Baart, Minnesota, 1969					
<11	0	–	0	–	0	–	0	–
11-19	0	–	6	71	0	–	0	–
20-30	0	–	0	–	6	32	0	–
			Baart, Colorado, 1969					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	0	–	4	45	4	40	4	61
			Lee, Minnesota, 1969					
<11	0	–	0	–	0	–	0	–
11-19	0	–	1	39	0	–	0	–
20-30	0	–	4	23	7	33	1	18
			Lee, Colorado, 1969					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	2	29	3	25	6	30	1	9
			Marquis, Minnesota, 1969					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	0	–	4	55	8	43	0	–
			Marquis, Colorado, 1969					
<11	0	–	0	–	0	–	0	–
11-19	1	57	0	–	0	–	0	–
20-30	2	37	8	49	0	–	0	–
			Mindum, Minnesota, 1969					
<11	0	–	0	–	0	–	0	–
11-19	2	11	3	26	0	–	0	–
20-30	2	27	3	12	2	0	0	–

Appendix Table 1. (Continued)

	Rate of epidemic development (slope)							
	Slow <0.05		Moderate 0.05-0.105		Fast 0.106-0.199		Very fast 0.20-0.30	
Onset stage*	No. of epidemics	Mean % loss	No. of epidemics	Mean % loss	No. of epidemics	Mean % loss	No. of epidemics	Mean % loss
			Mindum, Colorado, 1969					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	1	8	9	14	2	30	0	–
			Purdue, Minnesota, 1969†					
<11	0	–	0	–	0	–	0	–
11-19	0	–	5	55	1	70	0	–
20-30	0	–	2	25	4	16	0	–
			Purdue, Colorado, 1969					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	0	–	8	50	6	37	0	–
			Baart, Minnesota, 1970					
<11	0	–	0	–	0	–	0	–
11-19	0	–	9	73	1	67	0	–
20-30	0	–	6	37	5	46	0	–
			Baart, Colorado, 1970					
<11	0	–	0	–	0	–	0	–
11-19	0	–	2	66	0	–	0	–
20-30	0	–	2	70	11	36	5	26
			Baart, Puerto Rico, 1970					
<11	0	–	4	98	0	–	0	–
11-19	2	17	7	84	0	–	0	–
20-30	6	15	1	54	2	61	0	–
			Lee, Minnesota, 1970					
<11	0	–	0	–	0	–	0	–
11-19	0	–	6	57	1	69	0	–
20-30	0	–	7	31	6	46	0	–
			Lee, Colorado, 1970					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	1	3	6	4	10	14	3	16
			Lee, Puerto Rico, 1970					
<11	4	75	5	92	4	92	1	94
11-19	6	18	0	–	0	–	0	–
20-30	3	17	0	–	0	–	0	–
			Marquis, Minnesota, 1970					
<11	0	–	0	–	0	–	0	–
11-19	0	–	10	66	0	–	0	–
20-30	0	–	5	29	5	43	0	–
			Marquis, Colorado, 1970					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	0	–	0	–	19	26	2	24

Appendix Table 1. (Continued)

	Rate of epidemic development (slope)							
	Slow <0.05		Moderate 0.05-0.105		Fast 0.106-0.199		Very fast 0.20-0.30	
Onset stage*	No. of epidemics	Mean % loss	No. of epidemics	Mean % loss	No. of epidemics	Mean % loss	No. of epidemics	Mean % loss
			Mindum, Minnesota, 1970					
<11	2	55	0	–	0	–	0	–
11-19	11	33	6	44	0	–	0	–
20-30	0	–	1	41	0	–	0	–
			Mindum, Colorado, 1970					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	0	–	6	15	13	18	1	45
			Purdue, Minnesota, 1970					
<11	0	–	0	–	0	–	0	–
11-19	4	52	6	78	0	–	0	–
20-30	0	–	7	26	3	39	0	–
			Purdue, Colorado, 1970					
<11	0	–	0	–	0	–	0	–
11-19	0	–	4	62	0	–	0	–
20-30	0	–	5	23	11	37	0	–
			Purdue, Puerto Rico, 1970					
<11	2	98	3	97	2	98	4	100
11-19	5	20	8	87	0	–	0	–
20-30	3	1	0	–	0	–	0	–
			Baart, Minnesota, 1971					
<11	0	–	0	–	0	–	0	–
11-19	0	–	1	70	0	–	0	–
20-30	0	–	3	49	3	39	1	28
			Baart, Colorado, 1971					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	6	4	0	–	1	18	4	21
			Lee, Minnesota, 1971					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	0	–	1	3	7	13	0	–
			Lee, Colorado, 1971					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	4	3	1	2	1	6	0	–
			Marquis, Minnesota, 1971					
<11	0	–	0	–	0	–	0	–
11-19	0	–	1	57	0	–	0	–
20-30	0	–	0	–	5	45	2	16
			Marquis, Colorado, 1971					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	2	11	5	15	0	–	0	–

Appendix Table 1. (Continued)

	Rate of epidemic development (slope)							
	Slow <0.05		Moderate 0.05-0.105		Fast 0.106-0.199		Very fast 0.20-0.30	
Onset stage*	No. of epidemics	Mean % loss	No. of epidemics	Mean % loss	No. of epidemics	Mean % loss	No. of epidemics	Mean % loss
			Mindum, Minnesota, 1971					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	1	9	2	28	4	11	2	22
			Mindum, Colorado, 1971					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	2	13	2	10	0	–	0	–
			Purdue, Minnesota, 1971					
<11	0	–	0	–	0	–	0	–
11-19	0	–	3	51	0	–	0	0
20-30	0	–	3	11	0	–	1	10
			Purdue, Colorado, 1971					
<11	0	–	0	–	0	–	0	–
11-19	0	–	0	–	0	–	0	–
20-30	0	–	0	–	3	13	2	13

*Onset stage based on the Romig scale (figures 2 and 3).
†Purdue 5481C-1-13-2